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Obstructive sleep apnea and anesthesia

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Obstructive sleep apnea and disordered sleep syndromes are common throughout the population. As obesity of the general population continues to increase so too does the incidence of obstructive sleep apnea, as the incidence of upper airway fat deposition increases with obesity and thus the caliber of the upper airway decreases. This disordered breathing and airway obstruction syndrome poses significant challenges for the management of intraoperative anesthesia and postoperative pain.

PHYSIOLOGY OF THE UPPER AIRWAY

Obstructive sleep apnea (OSA) and obstructive sleep hypopnea (OSH) represent two forms of the same disorder: Upper airway collapse during sleep. The normal cycle of breathing involves maintenance of airway patency with the pharyngeal dilator muscles, which stiffen during inspiration to prevent lateral pharyngeal wall collapse. These muscles act opposite the inspiratory forces generated by the diaphragm and muscles of inspiration in order to allow air entry into the trachea. By contracting and stiffening the lateral pharyngeal walls, as well as by pulling the base of the tongue forward (ventrally), air is able to pass through the retropharyngeal area into the tracheal. The inspiratory force generated by inspiration is gauged by airway-pressure receptors in the pharynx. These negative pressure receptors in turn allow precise tensioning and control of the pharyngeal dilator muscles, thus preserving airway patency during inspiration. These negative pressure airway receptors can be blocked by pharyngeal local anesthesia[1,2].

OSA is common in obesity due to the propensity of fat deposition in the pharynx, resulting in pressure on the upper airway to close. The narrowing of the pharyngeal orifice makes closure of the pharynx during negative pressure inspiration much easier, and the pharyngeal dilator musculature cannot offset the negative pressure developed by diaphragmatic contraction. The deposition of fat along the lateral walls of the pharynx and in the submandibular region gives the obese patient submandibular jowls; the appearance of these jowls may harbinger the presence of OSA in an undiagnosed patient. The increased fat deposition in the obese patient to the nasopharynx and laryngopharynx, in the uvula, tonsils, tonsillar pillars, tongue, aryepiglottic folds, and the lateral pharyngeal walls, all result in narrowing of the pharynx and the increased likelihood that relaxation of the pharyngeal dilator muscles will result in collapse of the soft-walled pharyngeal airway[3].

DEFINITION OF OBSTRUCTIVE SLEEP APNEA (OSA) AND OBSTRUCTIVE SLEEP HYPOPNEA (OSH)

The diagnosis of OSA and OSH are based on criteria established form sleep studies. The diagnosis of OSA is distinct from central sleep apnea, although the two disorders may occur in the same patient. For purposes of this discussion, central sleep apnea will not be discussed.

No airflow for 10 seconds in the face of continued ventilatory effort for at least five times per hour of sleep is the definition of OSA. These events are usually associated with a decrease in arterial blood oxygen saturation of at least 4%. OSH is defined as a decrease of more than 50% in airflow, without a complete absence of airflow, for 10 seconds or more at least 15 times per hour of sleep. OSH may also be accompanied by a decrease in arterial oxygen saturation of 4% or more. Both OSA and OSH disrupt the normal patterns of sleep and result in arousal events, leading to other symptoms and consequences[4].

Symptomalogy of obstructive sleep apnea and obstructive sleep hypopnea

The pre-anesthetic interview may be the first instance in which a patient is asked about the characteristics and hygiene of sleep. The classic patient is an obese male with

pronounced jowls, who may be initially examined closely for potential difficulties with intubation. The majority of sleep apnea patients snore and this history is frequently elicited when the pre-anesthetic interview is conducted with a sleep partner present. The presence of obesity and snoring, particularly when the sleep partner describes periods of apnea, should alert the anesthesiologist to the potential presence of sleep apnea. Other symptoms of OSA include daytime somnolence, enuresis, pulmonary and systemic hypertension, coronary artery disease and myocardial infarct. Patients with OSA are also at risk for fatal arrhythmias during sleep as well as sudden death during sleep.

The physical characteristic of these patients frequently demonstrate an increased Body-Mass Index (BMI, defined as weight [kg] divided by height squared [in meters], i.e. having the units of kg/m^2) of $\geq 25\ kg/m^2$. In addition, the presence of lateral pharyngeal fat deposition, as described above, results in an exaggerated neck circumference, usually ≥ 17 inches (≥43 cm) for males or ≥ 16 inches (≥ 41 cm) for females[5]. Examination of the airway itself will generally reveal enlarged tonsils, prominent tonsillar pillars, enlarged nasal turbinates, possibly a narrowed maxilla or mandible, retrognathia, pronounced tongue and enlarged adenoids. These conditions are usually incorporated into the Mallimpati score used by anesthesiologists to predict difficulty with intubation. A high Mallimpati score, with anticipated intubation difficulty, should alert the anesthesiologist and perioperative team to the high potential for OSA or OSH in the patient. Indeed, Benumof advises that any patient with a difficult intubation should be considered to have sleep apnea until proven otherwise by appropriate studies[6].

DIAGNOSIS AND TREATMENT OF OSA AND OSH

Clinical suspicion, based on physical characteristic and history obtained from the patient and sleep partner, do not diagnose OSA or OSH, although in some instances the historical data may be virtually conclusive. For instance, the patient whose sleep partner reports snoring and episodes of apnea and arousal may be considered to have OSA, although the severity of the OSA may need documentation. Diagnosis of sleep apnea should result in interventions to lessen or eliminate apneic episodes and allow the patient to have normal sleep patterns.

A formal sleep study involves specific protocols and monitoring in order to diagnose and quantify OSA and OSH. Monitoring in a soundproof room during uninterrupted sleep is optimal for a diagnosis. Monitoring should include an electroencephalogram (to identify the stage of sleep and arousal events), an electrooculogram (to identify non-REM and REM sleep stages), oral and nasal airflow sensors and capnography (for airflow documentation), monitoring for snoring or snoring noises, esophageal and chest and abdominal movement monitors (to document respiratory efforts), oximetry, electrocardiography, submental and extremity electromyography (to monitor for pharyngeal dilator muscle activity and extremity movements), and occasionally invasive systemic and pulmonary pressure monitoring. This extensive monitoring will allow the precise diagnosis and severity of OSA and/or OSH to be recorded.

Polysomnography (sleep study) results are reported in terms of the number of apneic and hypopneic episodes per hours as well as the number of arousals per hour. The apneic and hypopneic episodes per hour are combined into the Apnea-Hypopnea Index (AHI); a value of 6-20 indicates mild OSA, moderate OSA is defined as an AHI value of 21-50, and > 50 AHI denotes severe OSA. The total number of times a patient arouses per hour is reported as the Total Arousal Index (TAI), and a combination of the TAI and AHI yields the Respiratory Disturbance Index (RDI). Central apneas (i.e., no airflow and no respiratory effort) are generally reported separately on the polysomnography report. Of interest, morbidly obese patients with documented OSA had central apneas in only 5.8% of the events reported in the AHI, demonstrating that the preponderance of apneas occurring in morbid obesity are related to upper airway obstruction[7].

SLEEP HYGIENE AND OSA

Normal adult sleep has two broad phases: REM (rapid eye movement) and NREM (non-rapid eye movement) sleep. NREM is further divided in to four stages, each signifying a progressively deeper stage of sleep. As NREM phase of sleep progresses, the electroencephalography of the subject progressively slows. The stage 3 and 4 «deep» stages of NREM sleep are thought to represent a restorative period of sleep. REM sleep results in a generalized loss of muscle tone and is when dreaming occurs.

NREM sleep causes the rhythmic activity of the upper airway dilator muscles to diminish and thus increases the upper airway resistance to inspiration. REM sleep causes upper airway muscle relaxation as a consequence of generalized muscle relaxation, and this disappearance of upper airway muscle activity can result in a large increase in airway resistance[8]. The near or complete occlusion of the upper airway creates a negative pressure in the pharynx during inspiration, potentially resulting in pharyngeal collapse and thus OSA. Partial collapse of the airway during this cycle of inspiration and increased upper airway resistance can result in fluttering of the pharyngeal walls and soft palate, thus causing snoring. Indeed, the sound intensity of snoring is well correlated with the severity of OSA and OSH, and the cessation of snoring in this setting is the result of total airway obstruction and OSA[4].

OBSTRUCTIVE SLEEP APNEA AND ANESTHESIA

The presence of OSA in the patient presenting for surgery results in several considerations for the anesthesiologist. First and foremost, an examination of the airway frequently finds the potential difficult intubation, a consequence of the patient's narrowed upper airway and fat deposition causing macroglossia and airway obstruction. Care must be taken to adequately evaluate the airway and anticipate the need for a fiberoptic awake intubation if laryngoscopy is not adequate to expose the glottis. Alternatively, newer methods of airway management, such as a Bullard laryngoscope, an intubating laryngeal mask airway, or other adjuvant laryngoscopic instruments may be used to accomplish intubation, if needed.

Maintenance of anesthesia depends in large measure on the type of surgical procedure to be performed. However, some caveats need to be observed. Firstly, OSA patients are usually obese; with obesity, a decreased functional residual capacity may be present owing to the restrictive nature of the patient's body habitus to chest expansion. Secondly, obesity may preclude use of spontaneous ventilation techniques due to the patient's inability to maintain spontaneous respiration in the face of the abdominal weight pressing of the diaphragm. Thirdly, pain control and extubation need to be carefully planned in order to allow maintenance of airway after muscle relaxant reversal and narcotic therapy for adequate pain control. An optimal anesthetic in the obese patient may necessitate aggressive regional anesthesia techniques, if possible, to diminish the need for post operative narcotic analgesia.

The use of narcotics in OSA patients is controversial. Some authors argue that minimal narcotics should be used, as OSA patients may be more susceptible to morbidity associated with airway collapse if their ventilatory drive is diminished with narcotic therapy. One recent retrospective review found that unanticipated complications, necessitating hospital admission, in outpatients with sleep apnea were not more frequent with longer acting narcotics than with short duration narcotics[9]. Whether patients with OSA are suitable for patient controlled analgesia (PCA) remains to be determined; however, it appears that OSA patients do not have an exaggerated response to narcotic medication.

Patients with diagnosed OSA are frequently treated with continuous positive airway pressure (CPAP) during sleep to alleviate OSA symptomology and OSA-induced changes in physiology. However, use of CPAP in patients with suspected but undiagnosed OSA may not be helpful. Patients frequently require a period of adjustment in order for CPAP to become effective. Thus *de novo* use of CPAP in patients with undiagnosed (or untreated) OSA may not result in effective post operative therapy; mask-fitting issues, the need for airway gas humidification, and establishment of optimal pressure settings may render CPAP unacceptable or ineffective for patients initiated to the therapy perioperatively[10]. Of course, patients treated with CPAP should have this therapy available in the post operative period, with the settings and equipment available and trained personnel caring for the patient to optimize therapy. It is reasonable to assume that these patients will be less susceptible to post operative complications of OSA, but this hypothesis has yet to be tested in a formal study.

The suitability of OSA patients to undergo outpatient surgery is frequently questioned. Sabers et al.'s[9] retrospective study of outpatients admitted for complications revealed a low unanticipated admission rate, suggesting that OSA-diagnosed patients were no more likely to have complications resulting in unplanned admissions than non-OSA patients. Thus, while OSA patients are more likely to require endotracheal intubation, in this study OSA patients could be treated as outpatients without a significant increase in morbidity.

POST OPERATIVE CARE OF THE OSA PATIENT

In the early post operative period, sleep disorders frequently occur, as patients are receiving analgesic therapy as well as being affected by the residual effects of general anesthesia. In addition, necessary nursing activities throughout the night frequently contribute to poor sleep hygiene. When OSA patients experience disorders in sleep cycle and poor sleep hygiene, REM sleep rebound and the accumulated effects of sleep deprivation may become a factor contributing to exacerbation of OSA[6]. It is because of these concerns the advisability of outpatient procedures in OSA patients has been questioned. Despite Sabers et al.'s data, no long-term information is available on recovery in OSA patients.

SUMMARY

Patients with obstructive sleep apnea and obstructive sleep hypopnea present many challenges to anesthesiologists. A review of the anatomy and physiology involved in this disorder reveals that the obese and morbidly obese are particularly prone to these disorders. A host of physiologic changes and symptoms accompany these syndromes which are typically not addressed in normal physician-patient interactions, yet anesthesiologists frequently elicit these symptoms and suspect OSA/OSH on the bases of airway examination and sleep history. Care must be taken in the management of anesthetic induction, intubation, and anesthetic maintenance of these patients, with particular attention to titration of neuromuscular relaxation and analgesic use. Perioperative and post operative management

should ideally include CPAP therapy in those with diagnosed OSA/OSH, yet no studies have demonstrated reduced complications with the use of CPAP in the immediate post operative period. Outpatient procedures, while not proscribed, must be approached with caution in the OSA patient; with careful management, however, the OSA patient will not experience a complication rate higher than that of the non-OSA patient.

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